

decrease together as lactation progresses. The amount of carbohydrates is lower than in any other series of milk analyzed. The last two factors hardly require attention, but the excessive proteids should be recognized.

The management of this condition is difficult; while ordinary colostrum milk soon adjusts itself, the milk of prematurity persistently maintains a high percentage of proteids. It may be reduced by administering large quantities of water to the mother or by pumping the milk and diluting with milk-sugar solution. Exercise, upon which we ordinarily rely for diminishing the proteids, is out of the question during the period of childbed.

Even if our efforts were successful the milk presents different characteristics from that later in lactation, and cannot be administered in safety. Many premature infants could be saved if this were generally known and the gastro-intestinal functions guarded with the care they demand. Attacks of vomiting and looseness of the bowels, with curdy movements, may seem trifling, but they are unwarranted in these delicate patients, especially when it is realized that a toxic gastro-enteritis with high temperature and fatal issue may ensue.

In cases of prematurity, then, the mother's milk should not be offered, but a wet-nurse secured. Her infant must be healthy, full term, two weeks of age (and, better, a month), in order that the characters of the colostrum period may be lost, and nothing will better determine the quality of her milk than its chemical examination.

The mother's breasts in the meantime should be pumped and massaged, so that they will not dry up, but after the proper change in function will offer the proper food. The change from wet-nurse to mother, however, must be gradually and carefully managed.

To summarize feeding in this condition, our indication is to administer a weak, digestible food, guarding against overtaxing the stomach and intestines. These organs must be protected, even if temporarily the nutrition of the infant appears to suffer, their future education being relied upon to take care of a stronger diet.

A CONTRIBUTION TO THE STUDY OF FATTY INFILTRATION OF THE HEART SECONDARY TO "SUBPERICARDIAL OVER-FATNESS."

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In this paper it is intended to consider, principally, questions relating to fatty infiltration of the heart as met with in extreme obesity. I desire also to note briefly the more distinctive symptoms of pure fatty overgrowth (fat around the heart). This will, it is hoped, assist in the dis-

crimination of the cases in which fatty infiltration is superadded. A certain small proportion of the cases of fatty infiltration are associated with graver forms of myocardial degeneration, principally fibroid and fatty. As the result of considerable personal experience with cases of fatty overgrowth and fatty infiltration the belief that fatty degeneration is a rare sequel of the latter condition is amply confirmed. The view that "fatty deposits in and around the heart often cannot be distinguished from fatty degeneration," however, is incontrovertible. Conversely, I have met instances that presented undoubted evidences of "subpericardial over-fatness" combined with features that aroused suspicion of fatty infiltration and degeneration, in which, after the over-fatness was removed, the heart-sounds became of normal strength and clearness, the features pointing to myocardial involvement disappearing.

According to E. Leyden,¹ the cases of "fat-heart" are divisible into two subclasses: (a) fatty overgrowth and (b) fatty infiltration. By the former term is meant an excessive deposit upon and around the heart in the subpericardial space; the latter (fatty infiltration) refers to an infiltration or a dipping of fat between the muscle-fibres even to the endocardium. While accepting this general classification, which is both convenient and expressive from a clinical stand-point, I would suggest for the first subclass the term "subpericardial over-fatness," since all cases present clear evidences of general over-fatness, rarely of partial over-fatness, and there is slight, if any, implication of the myocardium in most instances. On the other hand, the form of fatty infiltration under consideration is clearly a rare condition, if we accept the not uncommon instances hinted at above in which the morbid process is limited to a thin layer of muscle-fibres situated directly beneath the epicardium, and one met in extreme obesity, as a rule. When the infiltration is confined to the superficial muscular bundles it is more than doubtful if the characteristic symptoms of fatty infiltration arise. Clinically, it is important to make a clear subdivision of "fat-heart" into two classes: the one, a large class, easy of recognition, in which there is an abnormally large deposit of fat beneath the epicardium; the other, a small group, not easily recognized, in which interfibrillar fat deposits occur, and of graver significance. It is with the view to the adoption of a term that is more nearly distinctive of the condition, both as to the position as well as the significance of the fat-deposit, that I venture to suggest "subpericardial over-fatness" to indicate the more common and less serious form of "fat-heart."

Fatty overgrowth was described by a few of the earlier writers,

¹ Schott. *British Medical Journal*, August 18, 1894.

² Berlin. *klin. Wochenschrift*, April 22, 1878.

among them Kirkringius and Bonet.¹ Nothing of importance, however, was added to our accurate knowledge of the subject prior to the middle of the present century. Moreover, it has attracted a remarkably small share of attention since that epoch-making period in myocardial affections.² Mere obesity (subpericardial fatness) does not imply fatty infiltration; but it may in a mechanical manner produce respiratory and circulatory disturbances, particularly venous stasis, as shown by cyanosis, a frequent, small pulse and dyspnoea, or a distinctly asthmatic form of breathing and sometimes cough. These symptoms are commonly provoked by undue muscular exercise, which readily induces heart-strain and embarrassment of the respirations in the obese. From the continuation of the operation of such influences, as in certain occupations, venous stasis may lead to so-called passive catarrh of the mucous membranes. Certain bronchial and gastric phenomenon with which we are acquainted now ensue. In subpericardial over-fatness the heart's weight and labor are obviously increased. The poisonous products of tissue-metabolism are not eliminated in the normal ratio. Indeed, in one of my cases, a female, aged forty-two years, presenting marked obesity, weight 225 pounds, the daily renal output was diminished one-half from the normal health standard. The amount of food ingested even under these circumstances was plus, and the case exhibited all of the typical phenomena of lithæmia, of which the obesity was probably the cause. Doubtless the degeneration (fatty) of the myocardium that is sometimes witnessed in long-standing cases is secondary to the changes that often occur in the vascular system (arterio-sclerosis). It is conceivable also that, owing to the mechanical obstruction to the cardiac movements in consequence of the presence of a copious deposit of fat in the mediastinum and beneath the epicardium, the heart action may be totally and suddenly arrested as the result of violent exertion.

Morgagni³ narrates a case of a corpulent man, aged fifty-eight years, who had been suffering three years prior to death from attacks of angina pectoris. The autopsy revealed a large deposit of fat in the mediastinum and subpericardial areas. The heart was of normal size and apparently quite sound. He expressed the opinion that the cause of death was the enormous fat deposit which obstructed the current of blood in the heart and lungs. No mention, however, is made of the condition of the coronary vessels.

In two cases of subpericardial over-fatness, one reported by R. G. Hebb and the other by Hingston Fox, sudden death occurred. One

¹ Quoted by Alfred Stengel, Philadelphia Medical Journal, October 13, 1900.

² For the historical development of the subject the reader is referred to the excellent article of F. Forchheimer, M.D., AMERICAN JOURNAL OF THE MEDICAL SCIENCES, December, 1888.

³ Cor adipeplane tectum.

of these patients fell dead while running to catch a "bus;" the other, five minutes after being anesthetized. In both instances, however, rupture of the left ventricle was noted at autopsy. Hingston Fox's case, "from staining reactions of the muscle-fibres and their striations," showed no indication of either fatty degeneration or fatty infiltration of the heart-wall. In Hebb's case "fatty degeneration was absent." Such facts as these go to show that subpericardial over-fatness, looked upon usually as being free from immediate danger to life, may possess considerable medico-legal importance. Among the inevitable secondary effects occasioned by this interference of general fatty overgrowth with the heart mechanism and lung expansion are certain nutritive disturbances, the extent of which it is impossible to estimate with any degree of accuracy. Ordinarily, while at rest, the pulse is regular and moderately strong, although hard to reach in some cases. In the plethoric form of obesity, notwithstanding the presence of greatly thickened chest-walls, the great strength of the cardiac systole is often surprising, while the pulse is full and regular. On the other hand, particularly in the anæmic variety of obesity, it may be small, less tense (compressible), and slightly increased in frequency, but, as a rule, regular. A moderate grade of cardiac hypertrophy due to an increased demand upon the contractile energy of the heart is sometimes observed, but in many instances it is not found present. Says Kennedy,¹ *cardiac hypertrophy* (italics mine) "is not to be wondered at, for it would be strange indeed if, with an addition of from three to six ounces of fat placed on the organ, it would continue to carry on the same degree of action as in the normal state." It is often impossible to determine the size of the organ during life, owing to the overlying fat deposit. Enlargement is more common, for obvious reasons, in cases in which dilatation ensues in consequence of fatty infiltration. Not a few of the subjects of subpericardial over-fatness lead active, useful, and energetic lives.

It is impossible in the present state of knowledge to establish precisely the relation of this condition to fatty infiltration and myocardial degeneration, either fibroid or fatty. There is probably little convincing evidence to support the current view that subpericardial over-fatness is commonly, or even in any considerable percentage of cases, followed by fatty infiltration. According to my observation, without exception fatty infiltration was secondary to the pronounced form of anæmic obesity. Out of 103 cases, lasting for periods ranging from a few to many years, of extreme polysarcia that have occurred in my experience only five cases gave convincing proof of the existence of fatty infiltration. It has been reported, though rarely, in moderately corpulent

¹ Observations on Fatty Heart; Essay, 1880.

persons; but in such the symptoms are probably dependent upon a true fatty change due to causes other than fatty infiltration. It is in these cases that the renal output is markedly deficient. Again, the question how long subpericardial over-fatness continues as such before it terminates in fatty infiltration, in cases in which this sequel occurs, is unknown; but the usual period must, from personal knowledge, be measured by decades and not by years.

Rarely, however, we meet with exceptions to the rule. The clinical indications of fatty infiltration then appear early, and an alarming condition may soon be presented. The advisability and even the clinical importance of clearly differentiating mediastinal and extra-cardiac conditions from those that distinctly impair its contractile power, as, for example, fatty infiltration, must be obvious. In most instances, at least, the process of fatty infiltration reaches its highest development in the right ventricle, which in marked polysarcia may be completely covered by the deposit of fat, the left being rarely completely enveloped. The interfibrillar depositions are most marked near the surface of the organ. Fat infiltration exerts pressure upon and between the muscular fasciculi, inducing "atrophy and replacement" and embarrassing the action of the heart. This form of atrophy is secondary to and dependent upon the infiltration; but "some cases of primary atrophy of the muscle lead to the secondary development of fat in the connective tissue which is everywhere present and potentially fat-bearing."¹

A similar mode of origin of fatty infiltration is also exemplified in cases of pericarditis followed by adhesion of the visceral and parietal surfaces of the pericardium. It is conceivable that, as the result of mechanical compression by fat, the lumen of the coronary vessels is narrowed, with consequential defective nutritive supply to the muscle cells. Under these circumstances fatty metamorphosis or degeneration is the result, but this is probably an exceptional sequel.

In the cachexias of carcinoma and phthisis, as well as in the general senile atrophy, a moderate degree of subpericardial over-fatness, fatty infiltration, and true fatty change may all be found in association. In such instances the fatty infiltration may be, in part at least, secondary to atrophy of the muscle cells (part of a general atrophic process), and not the cause of the metamorphosis. At all events it may be assumed that it is only in the extreme grades of fatty infiltration that muscular atrophy from compression by infiltrated fat ("pressure atrophy") ensues.

I shall deal only with cases of fatty infiltration that are the consequence of extreme obesity combined with subpericardial over-fatness.

¹ R. Douglass Powell. Allbutt's System, vol. vi., 893, 894.

The etiology is obviously that of extreme general obesity, particularly the anæmic variety, however the latter may be produced. As a clinical entity, fatty infiltration is not sharply defined, and writers are not in perfect agreement as to the grouping of features on which to base even a reasonably assured diagnosis. Two conditions are among constant accompaniments, marked polysarcia and a feeble heart. It may be pointed out that in the majority of cases it is not until the symptoms of commencing dilatation, often as the result of sudden or unusual muscular exercise or a profound shock, develop that we are warranted in entertaining the belief that fatty infiltration has supervened. The more characteristic clinical indications are urgent dyspnoea upon muscular exercise, præcordial discomfort, pain under the sternum, angina pectoris, cardiac palpitation, arrhythmia, syncope, vertigo, and cyanosis. Various forms and grades of intensity of cardiac arrhythmia are commonly present.

Slight irregularities in which the pulse-waves occasionally vary in size and tension, or an occasional intermittence followed by a brief pause, are not indicative of fatty infiltration; at all events, they are not unfavorable symptoms. I have repeatedly observed slight intermittence and arrhythmia disappear as the result of treatment directed to the over-fatness. On the other hand, marked and constant disturbance of the cardiac rhythm is symptomatic of fatty infiltration and irremovable, although marked improvement may follow an appropriate course of treatment. Kisch¹ pertinently remarks: "Complete irregularity, *delirium cordis*, is to be regarded as a sign of grave disturbance of the heart mechanism which can never be removed, and is sometimes premonitory of sudden death." Cardiac arrhythmia is not, however, peculiar to fatty infiltration. Moreover, there are latent instances in which the pulse is full and strong and not increased in frequency. In a strongly suspicious case under my care at present the pulse is large, regular, and of high tension. With the appearance of ensuing cardiac dilatation the pulse-tension falls and the frequency of the heart's action increases. Irregularity also supervenes. Thus the transition of the disease from the earlier to the later stages may be noted, and these changes that occur in the development of the disease may serve to explain the differences of opinion among clinical observers as to the rate and other peculiarities of the pulse.

Henry Kennedy² bases his diagnosis upon a large, full pulse, not increased in frequency, an enlarged area of heart dulness; possibly a soft, systolic murmur over the base.

Bronchial asthma or an asthmatic type of breathing after a full meal, or in the absence of any exciting cause, is frequently observed in fatty

¹ Berliner klinische Wochenschrift, March 18, 1895.

² Loc. cit.

infiltration. A passive (hydrostatic) bronchitis, with cough and expectoration, is superadded in most cases. Angina pectoris is a recognized symptom. From personal experience, the apprehension of arterial disease, particularly sclerosis of the coronaries, is warranted in the majority of the cases that exhibit angina pectoris. Again, in the tabulated list of cases appended below arterio-sclerosis was present in two of the three fatal instances, but no particular mention of the condition of the coronary arteries was made in the autopsy notes. It may be pointed out here that in none of my cases was aortic regurgitation or adherent pericardium (so far as could be determined) in association, but in two of the five cases reported there were evidences of arterio-sclerosis. In Case I., in which severe attacks of angina still occur, the vasomotor apparatus is greatly disturbed during the seizures. I have had an opportunity to examine this case immediately after the paroxysm. So soon as the pain is over gaseous eructations occur; the heart's action, which is at all times markedly irregular, is unchanged, although the pulse at the wrist becomes almost imperceptible for a short period and dyspnoea is also temporarily aggravated.

The above symptom-group includes the most characteristic features, while other and more unusual manifestations are pointed out in the notes of the subjoined cases. The more or less characteristic features of fatty infiltration may become characterized suddenly, contrary to the general rule, in the course of subpericardial over-fatness which has lasted for a long period of time. Again, the turning-point in such instances may take place at a comparatively early period of life, as mentioned above, and the approximate cause may be a single systemic shock (as childbirth) or repeated shocks, such as occurred recently in a case under my immediate observation.

The notes follow:

CASE I.—Mrs. W. H. S., aged forty years, came under observation September 9, 1900. Menstruation had been regular, although somewhat scanty. The patient had always been stout, and now weighs 220 pounds; height, 5 feet 8 inches. Two children born, the second one when patient was thirty-eight years of age. The father died of Bright's disease and one brother of phthisis; otherwise, family history was uneventful. The patient had enjoyed good health until four years previously, when she had a severe attack of influenza. One year later sustained a severe shock through death of a brother, and after the lapse of another year a second shock due to the prolonged second childbirth, which was attended with much hemorrhage. Following up this event exhaustion was extreme and life despaired of; but she slowly recovered, although remaining in a highly nervous condition, with marked arrhythmia and dyspnoea on slight exertion. The death of her child, at ten months of age, plunged her into profound grief, aggravating at the same time the symptoms mentioned before. Four months later I was called. Patient now suffering from cough, asth-

matic breathing, and *delirium cordis*; the pulse small, soft, compressible, and too irregular and intermittent to be recorded at the wrist. The skin surface was pale; no cardiac impulse visible; the heart could not be satisfactorily outlined by percussion, although some degree of enlargement was evident; palpation detected an exceedingly feeble, irregular, and at times intermittent impulse. On auscultation no murmur could be heard, but sibilant râles over the bronchi; a low-toned, wheezing sound on expiration was audible. The urine contained a trace of albumin, and the acidity was slightly increased. Patient extremely nervous and often weeps while in ordinary conversation; slight twitching of the muscles of the face observed almost constantly; has headaches at frequent intervals. May not some of these nervous symptoms be ascribed to uræmic intoxication? is a pertinent question. I may add that similar nervous symptoms, although less marked in two, were also observed in all of the remaining cases herein recorded. This patient has been troubled with cough and a mucoid expectoration after having lain in bed for a few hours at perfect rest. The cause of these symptoms is probably a hydrostatic bronchitis due to increased feebleness of the action of the heart while the patient is at absolute rest.

The patient has had occasional anginoid attacks during the past four months, and more recently has complained almost constantly of pain behind the lower portion of the sternum; exacerbations of precordial pains also occur, during which she cannot lie down; has great trouble in breathing; awful forebodings; is restless, and the skin grows pale and moist. Stimulants afford relief at times, but the worst attacks demand morphine hypodermatically.

Present Condition (November 29, 1900). The attacks of anginiform pains are improving and the heart's action is somewhat more regular and slightly stronger.

December 18th. Has had fresh exacerbations as the result of over-exertion and severe angina.

There came under my notice recently a case in which the opinion that fatty change in the heart fibres was associated with extreme fatty overgrowth had been long entertained. The notes of Case II. are as follows:

CASE II.—Mrs. J. B., aged fifty years, of temperate habits; has seven children; is still menstruating regularly, although the flow has long since been somewhat scanty. The father and one sister are moderately obese; mother died of phthisis and a sister suffered in early life from acute rheumatism. The patient has had the usual childish affections, and two years previous to consulting me had an attack of influenza. Patient had become progressively stouter from the time she began to bear children up to the date of my first visit to her, at which time the bodily weight was 310½ pounds. Previous to her marriage had been moderately fleshy. For ten years there had been marked dyspnoea and palpitation on exertion, and when I first saw her locomotion was wellnigh impossible, efforts at walking being quickly followed by extreme dyspnoea, cyanosis, and by a feeling of utter exhaustion. Attacks of cardiac asthma rarely occurred, but there were no anginiform pains.

Physical examination revealed a body of enormous proportions. The impulse was feeble and diffuse to palpation. The first sound of the heart was scarcely audible and its action was both intermittent and irregular; no murmurs were present. The limits of cardiac dulness could not be made out satisfactorily, although the left boundary line was found to approach the anterior axillary line, while slight abnormal extension to the right was noted. The pulse was small, compressible, and at times intermittent (like the cardiac action). As the result of an appropriate regimen, including massage until physical exercise could be adopted, the bodily weight was reduced to 185 pounds. At this time, fourteen months after the commencement of the treatment, the patient could readily walk four miles daily, and the heart-sounds were clear and almost regular. The enlargement of the left ventricle was now slight, and the diminution in the apparent size of the heart was due largely to the removal of the subpericardial layer of fat.

It may be questioned whether this case should be classified as one of fatty infiltration in view of the favorable issue that resulted from treatment and a proper regimen. It may, however, be admitted that slight and even moderately extensive fatty infiltration is rarely curable. On the other hand, the outcome thoroughly disproves the existence of fatty change in the muscle-fibres of the myocardium, or fatty degeneration, for when once this grave disease is established it progresses to a fatal ending.

CASE III.—A. P. B., aged forty-seven years; occupation, clerk; married; consulted me May 22, 1896. Patient hereditarily predisposed to insanity and phthisis. As a young man, was perfectly healthy until the age of twenty-two years, when he began to suffer from muscular rheumatism, and on two occasions since then has had lumbago. One year prior to consulting me had sciatica, confining him to the house for two days only. Had been growing stouter in a slow and insidious manner for fifteen years; this is attributable to the habitual use of malt and spirituous liquors coupled with the lack of muscular exercise. Present weight, 210 pounds; height, 5 feet 6 inches; user of tobacco in excess. Two years previous to his first visit he began to experience peculiar sensations and discomfort in the præcordia, sometimes inducing a sense of impending death. Muscular exertion was followed immediately by moderate dyspnoea and by pain in the præcordial region, which became worse if exercise was continued, and finally he would be compelled to desist. For at least ten years has had slight winter cough and distinctly asthmatic breathing at intervals. Dyspnoea, cough, and cyanosis have been for a long time, and still are, conspicuous features.

Physical Signs. Inspection reveals corpulent frame, abdomen unusually prominent and pendent, lung expansion somewhat restricted, and the apex-beat of the heart invisible. To palpation the tactile fremitus was normal and the cardiac impulse quite feeble and irregular; the pulse-waves were small, decidedly irregular, and slightly increased in frequency; percussion shows moderate enlargement of the heart, more especially toward the left; auscultation reveals a few dry râles over

larger bronchi; sibilant and subcrepitant râles over bases posteriorly; the heart-sounds are feeble and irregular; there are no cardiac murmurs, but the aortic second sound is slightly accentuated.

The treatment embraced the withdrawal of all the alcoholic stimulants and decided restriction in the use of cigars. Methodical and persistent exercise, including light gymnastics, was enjoined, commencing with a minimum amount and gradually increasing to three and finally to four miles daily. The regimen also included a dietary calculated to overcome the obesity. Improvement in the local manifestations was soon noted, the bodily vigor and endurance were greatly increased, and the bodily weight reduced to 185 pounds. Three years later, on July 29, 1899, the following note was made: Patient still suffers occasionally from slight attacks of angina and from anginoid pains in the intervals. The pains are aggravated by undue exertion, marked nervous disturbance, or acute indigestion. The heart's action is markedly improved in consequence of treatment, although still irregular and rarely intermittent.

The history of rheumatism in the family as well as in the patient during early life at first gave rise to the thought that the moderate grade of arterio-sclerosis might be of rheumatic origin. The happy effects of treatment upon the arrhythmia, anginiform pains, and the general condition of the patient, however, contradicted this view and emphasized the etiological importance of the habitual use of alcohol and excessive smoking in the case. The action of alcohol in the production of obesity is well known, and the same is true of its causative influence in arterio-sclerosis. It is quite probable, at all events, that the discontinuance of these toxic substances, particularly the alcoholics, had most to do with the rapid and favorable modification of the local and general condition of the patient.

CASE IV.—J. V., aged fifty-two years, American by birth, public official, consulted me on July 10, 1900. Patient weighed 225 pounds, five feet seven inches tall, and his habits were nocturnal. Has been an excessive smoker for many years. There is a clear history of rheumatism and gout in maternal ancestry. The personal history furnishes no points of value up to ten years ago, when he began to grow stout, probably in consequence of a change from an active to a sedentary, irregular life combined with a rather free use of alcoholic intoxicants. No acute illness, although dyspnoëic on exertion and asthmatic at intervals, with occasional vertigo, until the onset of the complaint for which he applied for treatment. The exciting cause of the latter was probably heat-exhaustion and the improper use of cold water and ice during its treatment. Dyspnoëa quickly ensued, accompanied by marked cyanosis and severe anginoid pains in the region of the heart, radiating to the neck and arms, particularly the left. The heart's action became wholly irregular, both in time and volume, and the accessible arteries were somewhat hardened; vertigo and syncope were also prominent features. The physical examination revealed a soft, low-pitched, systolic murmur near the base, with increased loudness of the aortic and pulmonary artery second sound. Dilatation of the ventricles could not be shown

by percussion, owing to the over-fatness. Although a marked improvement resulted from the treatment, which was conducted on lines similar to those in Case III., the symptoms have continued in a milder form.

The predisposing causes in this and the previous case were almost identical. The urgent symptoms (angina, *delirium cordis*, etc.) were ascribable to the acute development of heat-exhaustion, or, since they did not supervene until after the wrong use of hydrotherapy, they may have been excited by it. This is a second instance in which the evidences of infiltration following overgrowth were developed acutely. (See Case I.) These cases go to show that fatty infiltration may be latent, up to a given point at least.

CASE V.—A. B. M., aged thirty-six years; no occupation, single, weight 237 pounds (in bath); first applied for treatment February 1, 1899. Is a habitual drinker of intoxicants and particularly "spirits." The mother is rheumatic and there is a family predisposition to obesity. After suffering from childish diseases patient sustained a fall at the age of twelve years; this caused concussion of the brain, confining him to bed for three months. Patient has been stout from the time of his earliest recollection, but in recent years has suffered from vertigo and occasionally syncope, dyspnoea, præcordial discomfort, and anginoid pains, particularly on muscular exercise. The palpable arteries are not hardened; the pulse is soft. On auscultating the heart an intermittence which occurred every third or fourth beat and an occasional false intermittence were noted. Slight cardiac enlargement was elicited by percussion. A faint systolic murmur was heard at the base, though not constantly. At intervals *delirium cordis* was observed. The rhythm was less intermittent and less irregular by far in the standing posture than in the sitting or the recumbent posture. True angina pectoris has not developed. Appropriate treatment for the obesity and the use of cardiac stimulants resulted in a loss of weight amounting to thirty-one pounds and an improvement in the more conspicuous features, particularly the arrhythmia, dyspnoea, and cyanosis. Patient has not been under observation during the past year, although still living.

The cases briefly reported above show pretty clearly that the transition from subpericardial over-fatness to fatty infiltration may be, so far as the symptomatic indications go, abrupt, on the one hand, and too insidious to fix the date of onset of the latter complaint, on the other hand. In reviewing my cases it is seen that the most conspicuous symptoms were marked dyspnoea, syncope, and utter exhaustion on muscular exercise, with cyanosis, præcordial distress, anginiform pains at frequent intervals, and less commonly true angina, well-marked arrhythmia, and even *delirium cordis*; also asthmatic breathing and certain nervous phenomena, particularly emotional disturbance and mental apprehension. A basal systolic murmur was present in two cases and cardiac enlargement was detectable in four. The urine con-

tained a trace of albumin in one (Case I.). In cases in which a murmur over the aortic area was present vertigo and syncope were annoying symptoms. It may be stated here that a basic systolic murmur is also rarely heard in cases of subpericardial over-fatness combined with vigorous cardiac contractions.

With reference to the question of diagnosis in this complaint it must be observed that neither the subjective symptoms nor the objective signs alone or in combination are conclusive, and partly for the reason that they vary considerably in different cases. The presence of the causative condition, extreme pericardial over-fatness, is all-important, and at once gives strength of probability to a diagnosis as one after another of the more characteristic features recited above put in an appearance. An assured recognition of the disease is finally arrived at only after the closest scrutiny of all the symptoms and physical signs coupled with a judicious balancing of the data entering into the previous history of the patient. Should any of the primary affections, other than extreme obesity, that are liable to lead to fatty infiltration be present, this fact would clearly bear upon the case, and the diagnosis of the special form of fatty infiltration under consideration would be precluded. Mere feebleness of the heart is not of great diagnostic value, taken alone. We see this in a variety of conditions and diseases. I desire also to reiterate the fact that slight arrhythmia is not of any value for diagnosis in fatty infiltration.

I have appended a tabulated list of cases of fatty infiltration (unassociated with degeneration of the heart). J. Daland,¹ J. H. Crocker² and others have also reported cases, but inasmuch as in all of them fatty degeneration was combined with fatty infiltration they are not included in my table.

¹ Transactions of the Pathological Society of Philadelphia, 1893 and 1894, p. 92.

² London Lancet, January 4, 1890.

No.	Name of reporter.	Age and sex.	Weight.	Symptoms.	Arterio-sclerosis.	Mode of death.	Condition of heart.	Remarks.
1	Satterthwaite ¹	M. 70	357 lbs.	Marked dyspnea; pulse feeble and intermittent; impulse of heart feeble.	Slight, secondary to nephritis.			Lost 40 lbs.; marked improvement.
2	Satterthwaite	F. 65	181 lbs.	Color dusky, skin bathed in perspiration; pulse 100 to 140; weak impulse at apex; heart enlarged; no murmur.				Lost 10 lbs.; felt much improved.
3	Satterthwaite	M. ..	227 lbs.	Unusually subject to giddiness of head, acute gasconous dyspnea, and dyspnea; pulse 100; no intermission; apex-beat feeble, difficult to locate; heart enlarged.				Lost 20 lbs. in two months. General condition much better.
4	W. H. Thayer ²	M. 64	Completely built; weight not given.	Previous history was negative. Patient taken ill with weak heart and indigestion. On third day pseudo-epileptic attacks developed; dyspnea marked until five days prior to his death. Temperature 100°; no cardiac signs; temperature slightly elevated before death; pulse 100; settling patient up, pulse became irregular.		Rather sudden.	Microscopical examination of a portion of the wall of the left ventricle revealed no fatty degeneration, fat deposits and pericarditis were noted and the heart was dilated.	Lungs congested, also peritonitis.
5	David Hunter ³	F. 77	Quite obese; weight not stated.	Had suffered from chronic malaria and secondary dementia; was confined to her bed for a few weeks. On second day of her illness she had a swelling of her knee and leg, and dyspnea; cardiac action feeble for the last two years; was subject to swelling of feet and ankles and cyanosis, but no cardiac bruit detected; after three or four sleepless nights had dyspnea and unusually cyanosed. She died five days afterward.	Some (chronic) renal (nephritic).	Died rather suddenly (rupture of heart).	Right ventricle ruptured, due to fatty overgrowth, and great greenish, fatty, scum-like adherent pericardium.	Lost 10 lbs.
6	R. G. Hobbs ⁴	F. 62	Body adipose; weight not given.	Patient was undisturbed after a careful examination with negative results was made; five minutes after anesthesia had been caused patient became cyanotic and died after all means of resuscitation had failed.	Present.	Sudden.	General fatty infiltration of ventricle walls. (Autopsy).	A typical case of fat-heart.
7	O. Clemow ⁵	M. 50	229 lbs.	Patient had led an active life for several years and used malt liquors to excess. When first seen patient complained of dyspnea and difficulty in walking. On second day of illness he had a swelling of the right border extended about an inch to right of sternum; first sound muffled and indistinct at apex; no murmur anywhere; pulse 72, feeble.				Lungs healthy; patient became ill with hepatic congestion and loss of weight; when he recovered Oedema of the extremities and edema of the diaphragm was instituted with marked amelioration of the symptoms.

¹ Post-Graduate, New York, March, 1899.

² Brooklyn Med. Journ., February, 1890.

³ Med. Press and Circular, April 29, 1891.

⁴ Trans. Path. Soc. of London, Vol. XLIX, p. 33.

⁵ London Lancet, December 18, 1897.

REMARKS. Obviously, from these studies, pure fatty infiltration as a sequel of "subpericardial over-fatness" is an extremely rare condition.¹ Of the three fatal cases given in the table above one ended with great suddenness and the remaining two rather suddenly. It will be noted that in the case of sudden death arterio-sclerosis was associated, and in one of those that terminated rather suddenly the lesions of chronic interstitial nephritis were noted. It is evident that the arteries were also the seat of sclerotic change in the latter case. The comparative frequency of atheroma, particularly of the aortic arch and the coronaries, as an associated lesion in fatty infiltration is undoubted. Thus, Forchheimer noted it in thirty-nine out of 122 cases of fatty overgrowth gleaned from the literature prior to 1889. Of these, atheroma of the aorta occurred in twenty-one and similar lesions of the coronaries in fourteen cases. Kennedy² invites attention to the comparative rarity of ossific deposit on the valves, and this view, long since expressed, receives support from the post-mortem findings of the three cases given in the above table, since valve lesions were not noted in a single instance.

Von Stofella³ observes that when in subpericardial over-fatness the impulse is feeble and the first sound muffled and indistinct the aortic and pulmonary second sounds are of natural loudness, whereas in fatty infiltration the second sounds are feeble. If arterio-sclerosis be associated, then the second sounds may appear to be somewhat accentuated; this was true of Case IV. of my series. In Case III. slightly increased loudness of the aortic second sound was present in the absence of hardening of the peripheral arteries. The murmurs present in fatty infiltration have their usual seat in the aortic area, although they may also be heard in the mitral zone. When met at the base of the heart they are attended with troublesome cerebral symptoms, syncope and vertigo in particular. I have had no personal experience to confirm the view that this is also true of mitral murmurs in this affection. Murmurs are not constant in this disease, having been present, as previously stated, in but two of the cases reported above. They are not due to chronic valvulitis. Says Forchheimer,⁴ speaking of the bruit in fat-heart: "The bruit which is heard must, I take it, be due either to the irregular contraction of the muscular fibres of the heart or it must be hæmic in origin." The evidence is overwhelmingly against its being due to valvular lesions, since these are the exception in cases of fatty infiltration. Personal observation tends to confirm the belief that I have long held regarding this question; it is that the bruit heard in this affection may have different causes, many of which are as yet unknown. It can

¹ The writer would be indebted to anyone who will furnish additional cases.

² Loc. cit.

³ Wiener klinische Wochenschrift, 1893, vi.

⁴ Loc. cit.

scarcely be doubted, I think, that the murmur that appears in the advanced stages of the disease is at times due to relative incompetency, since many other indications of a dilated heart are in association. In some cases the murmur heard over the aortic area is attributable to an associated chronic nephritis, since the presence of a basic systolic murmur in the latter disease is a matter of common observation. Again, the bruit may be hæmic in origin or due to an abnormal, relaxed state of the heart-muscle or to weakness or insufficiency of the papillary muscles.¹ The moderate degree of enlargement in four of my cases showed itself by an increase of dulness more to the left than to the right and more in the transverse than in the vertical direction. It is probably occasioned principally by dilatation which may be preceded by and associated with some hypertrophy caused by the over-fatness. The rôle played by the infiltrated fat as well as the abnormally great subpericardial fat deposit must also be taken into account. It is always exceedingly difficult and sometimes impossible to establish the boundary lines of dulness by percussion, owing to the extreme corpulency. Not to be neglected in the treatment of these cases are measures directed to the removal of the over-fatness, the accomplishment of which enables us not only to determine accurately the size of the heart, but also to improve both the cardiac action and general condition of the patient.

A fatal termination is often due to spontaneous rupture of the heart, as occurred in all of the cases given in the above list that came to necropsy. This accident, however, does not, as shown by the clinical notes of Cases IV. and V. in the above table, invariably cause sudden death.

MULTIPLE NEURITIS AND HÆMATOPORPHYRINURIA FOLLOWING THE PROLONGED INGESTION OF TRIONAL.

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THAT poisoning by trional is an exceedingly rare condition is readily borne out by the fact that it has been freely administered in thousands of cases in the past decade for insomnia, as a sedative, and as an antipyretic in a wide range of diseases, and is generally considered a reliable and effective remedy, without untoward effects. I have been impressed by the readiness with which patients acquire the trional habit and the difficulty of inducing them to abandon its use. Their attitude has, I think, often been determined by the physician who first

¹ See Schroetten, Ziemssen's Hand-book, vol. i. p. 25.